This Portion contains the following comment letters:

Local Organizations

Letter I	San Diego County Archaeological Society, Inc.	PR-52
Letter J	Scripps Mercy Hospital	PR-57
Letter K	Environmental Health Coalition	PR-58

San Diego County Archaeological Society, Inc.

Environmental Review Committee

25 September 2005

To: Mr. Steve Power, AICP
Environmental Projects Manager
City of Chula Vista
276 Fourth Avenue
Chula Vista, California 91910

Subject: Recirculated Draft Environmental Impact Report
Chula Vista General Plan Update

Dear Mr. Hellman:

I have reviewed the cultural resources aspects of the subject Recirculated DEIR on behalf of this committee of the San Diego County Archaeological Society.

Based on the information contained in the Recirculated DEIR, we have the following comments:

I-1 1. We urge the City to consider and recognize the industrial historic resources such as the Western Salt Works and the sites of the other operations mentioned in Section 5.4.1.2.a Such facilities are often overlooked, yet they form an important part of the City's development history.

I-2 2. We commend the City for its decision to participate in the Mills Act program. The benefits this program offers are a valuable component of a historic preservation program.

I-3 3. In Section 5.4.1.2, in the paragraph beginning "In 1885," the description of the five-acre lots refers to "streets 80 feet in width and a steam motor passing through the center." The meaning of this sentence is not clear.

I-4 4. In Section 5.4.3 of the DEIR, Policy EE 9.2 calls for supporting and encouraging the accessibility of the City's cultural resources. To accomplish this, for archaeological collections, the City should undertake research to determine where collections from previous CEQA-mandated projects are currently located, and their condition. Efforts should then be made to upgrade those collections, as necessary, and bring them into a qualified curation facility (see 10, below) It should be understood that, if a project relies upon previous work, the collections from that previous work must also be curated along with the new collections.

5. The first paragraph of Section 5.4.4, Threshold 1, states: "In open areas, there is the potential that future development, as permitted by the plan, could impact historic and prehistoric sites." This possibility does not exist solely in open areas. Portions of sites may exist under

PR-52

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RESPONSE

I-1 The dEIR evaluated cultural resources including industrial historic resources. Page 241 of the dEIR states that a cultural resource may: be the location of a prehistoric or historic occupation or activity; be a locale which has been, and often continues to be of religious, mythological, cultural, economic, and/or social importance to an identifiable ethnic group; be associated with events that have made a significant contribution to history or cultural heritage; be associated with the lives of important persons; embody the distinctive characteristics of a type, period, region, or method of construction; represent the work of an important creative individual; possess high artistic values; or yield information important in prehistory or history. These resources encompass industrial historic resources. Page 244 of the dEIR discusses the Western Salt Works which has been operating on the Chula Vista bay front since the beginning of the century. Compliance with the policies associated with Objectives LUT 12 and EE 9 and the mitigation measures identified on Page 253 and 254 of the dEIR would reduce the impact to cultural resources resulting from the adoption of the General Plan Update to below a level of significance.

I-2 These comments do not reflect on the adequacy of the EIR. The comment, however, will be forwarded it to the appropriate City Decision making body. Comment noted.

I-3 Comment noted. The sentence has been revised to provide the following clarification:

They began developing the area by subdividing a 5,000-acre portion into five-acre lots. The lots were separated with avenues and streets 80 feet in width and a steam motor passing through the center of the streets.

I-4 These comments do not reflect on the adequacy of the EIR. The comment, however, will be forwarded it to the appropriate City Decision making body.

San Diego County Archaeological Society, Inc.

Environmental Review Committee

25 September 2005

To: Mr. Steve Power, AICP
Environmental Projects Manager
City of Chula Vista
276 Fourth Avenue
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1. We urge the City to consider and recognize the industrial historic resources such as the Western Salt Works and the sites of the other operations mentioned in Section 5.4.1.2.a Such facilities are often overlooked, yet they form an important part of the City's development history.

2. We commend the City for its decision to participate in the Mills Act program. The benefits this program offers are a valuable component of a historic preservation program.

3. In Section 5.4.1.2, in the paragraph beginning "In 1885," the description of the five-acre lots refers to "streets 80 feet in width and a steam motor passing through the center." The meaning of this sentence is not clear.

4. In Section 5.4.3 of the DEIR, Policy EE 9.2 calls for supporting and encouraging the accessibility of the City's cultural resources. To accomplish this, for archaeological collections, the City should undertake research to determine where collections from previous CEQA-mandated projects are currently located, and their condition. Efforts should then be made to upgrade those collections, as necessary, and bring them into a qualified curation facility (see 10, below) It should be understood that, if a project relies upon previous work, the collections from that previous work must also be curated along with the new collections.

I-5 5. The first paragraph of Section 5.4.4, Threshold 1, states: "In open areas, there is the potential that future development, as permitted by the plan, could impact historic and prehistoric sites." This possibility does not exist solely in open areas. Portions of sites may exist under

PR-53

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RESPONSE

I-5 While it is true that Figure 5.4-1 addresses only the potential for prehistoric archaeological resources and not historic archaeological resources, mitigation measure 5.4-1 listed below was established to reduce impacts to historic resources resulting from the adoption of the General Plan Update to below a level of significance.

5.4-1 Implementation of Policies LUT 12.7 and EE 9.1 shall include the following measures:

1. Any future development project that has not been previously examined shall be subject to a cultural resource survey or review, to identify any specific resources that could be potentially affected by the proposed project.

2. In western Chula Vista, an archaeological survey shall be completed for any development project that includes previously undisturbed acreage and has not been previously examined or for which there is reason to expect a potentially significant historic or prehistoric archaeological resources, to identify any specific resources that could be potentially affected by the proposed project.

3. The City will promote maintenance, repair, stabilization, rehabilitation, restoration, and preservation of historical resources. Where these will be undertaken, they will be conducted in a manner consistent with the Secretary of the Interior's Standards for the Treatment of Historic Properties with Guidelines for Preserving, Rehabilitating, Restoring, and Reconstructing Historic Buildings.

4. Prior to the approval of any projects that propose to demolish or significantly alter a potentially significant historic resource, as defined pursuant to applicable state and federal laws, shall complete an historic survey report to determine potential historic significance. The determination of resource significance shall be made in accordance with CEQA Guidelines Section 15064.5 and the program established as a result of Policies LUT 12.3, 12.4, 12.7, and 12.11 and EE 9.1, and shall be completed to the satisfaction of the appropriate decision maker.

5. In the event that significant resources could be adversely affected by the proposed action, as established in Policy LUT 12.12, a conservation program shall be implemented in accordance with applicable state and federal laws, to the satisfaction of the appropriate decision maker. The conservation program shall be designed to reflect the reason that the identified resource is considered important. Where appropriate for a standing historic structure that will not be preserved in place, conservation can include documentation to Historic American Building Survey (HABS) standards and/or relocation. For archaeological remains, conservation of a resource for which preservation in place is not feasible would include the execution of a research design directed program of scientific data collection and analysis.

current development, such as streets or structures where extensive grading did not take place. This is particularly possible for historic archaeological resources, such as privies, and it especially applies in the western portion of the City. It is worth emphasizing that Figure 5.4-1 addresses only the potential for prehistoric archaeological resources, not historic archaeological resources.

I-6 6. In Section 5.4.5, Mitigation Measures 1 and 2 refer to archaeological surveys being required for previously-unsurveyed projects. Projects where surveys more than five years old are generally considered to require resurvey, whether or not positive results were obtained, and this standard should be reflected in this Section. Also, please note that in addition to a survey, records searches are necessary, and review of maps and aerial photographs can assist in identifying previous land uses and resources.

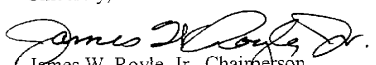
I-7 7. Mitigation Measure 4 refers to relocation of historic structures. It must be noted that relocation of a historic structure destroys some of the significance of the resource. While certainly preferable to demolition, it does not mitigate impacts to a level of insignificance. Therefore, where relocation is part of the "mitigation" for a project, the City will need to incorporate findings to substantiate why the impacts cannot be mitigated to the point where they are not significant.

I-8 8. In Mitigation Measure 5, for archaeological resources, archaeological monitoring may be an appropriate part of the mitigation measures for a particular project.

I-9 9. And also for Mitigation Measure 5, for archaeological resources, the DEIR must recognize that mitigation is not complete without curation of the resulting collections and associated records in a facility meeting the standards defined in *Guidelines for the Curation of Archaeological Collections*, dated May 7, 1993, and available from the California Office of Historic Preservation. Not curating collections is a violation of Section V of the Code of Conduct of the Register of Professional Archaeologists.

Thank you for providing this recirculated environmental document to SDCAS for our review and comment.

Sincerely,



James W. Royle, Jr., Chairperson
Environmental Review Committee

cc: SDCAS President
File

PR-54

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RESPONSE

I-6 Currently, it is not a General Plan policy to require re-surveying every five years. It is a policy to survey every property proposed for development that has not been surveyed. CEQA requires mitigation for any site that has been determined to be significant.

I-7 While it is true that relocation could destroy some of the significance of a historic resource, whether or not relocation is an appropriate mitigation varies from project to project. To determine this, Mitigation Measure 5.4.1 (4) requires a historic survey report to determine potential historic significance would be required prior to the approval of any projects that propose to demolish or significantly alter a potentially significant historic resource, as defined pursuant to applicable state and federal laws.

I-8 Comment noted. Archaeological monitoring may be part of the program established for a particular project, however, it is not in itself sufficient mitigation if the site is going to be destroyed.

I-9 Project level environmental analysis will address curation. This is not a General Plan issue.

"...existing residential neighborhoods." in the "Promenade Area":

TYPE	NAME	NO. OF UNITS
Condominiums	Holiday Gardens	164
Manufactured Homes	Terry's	196 (55+)
	Carillo's	125 (55+)
	Broadway	73 (55+)
	Bison	79 (55+)
	Mohawk	15 ("older person")
	Rose Arbor	120
	Caravan	42
	Trailer Villas	146
	Flamingo	61
Apartment complexes	Vistan	352
	South Bay Towers	132
	Pine Tree Manor	32
	Woodlawn Gardens	150
	Sunny Side Manor	32
	Vista Village	90
	Cambridge	41
	St. Thomas	77
	Park Regency	125
	Woodlawn	117
	Woodlawn 20	20
	Sunset 8	8
	Woodlawn Colonial	136
TOTAL		2,333

LOWER SWEETWATER
E STREET GATEWAY
DOWNTOWN THIRD AVENUE
H STREET GATEWAY
H STREET CORRIDOR
MID-THIRD AVENUE
MID-BROADWAY
FOURTH
FIFTH
THIRD
SECOND
FIRST
PLAZA BONITA
HILLTOP
COUNTRY CLUB
LAGOON
Note: Existing Land Use Colors are Screened Back
1 INCH = 1,800 FEET
CITY OF CHULA VISTA
ADVANCE PLANNING SECTION
CITY OF CHULA VISTA
NORTHWEST
GPU STEERING COMMITTEE
RECOMMENDED ALTERNATIVE
LEGEND
RESIDENTIAL
LOW
LOW MEDIUM
LOW MEDIUM VILLAGE
MEDIUM
MEDIUM HIGH
HIGH
URBAN CORE
COMMERCIAL
RETAIL
VISITOR
PROFESSIONAL & OFFICE
MIXED USE
MIXED USE COMMERCIAL
MIXED USE (OFFICE, RETAIL & MULTI-FAMILY)
MIXED USE TRANSIT FOCUS AREA (TFA)
INDUSTRIAL
LIMITED INDUSTRIAL
RESEARCH INDUSTRIAL
GENERAL INDUSTRIAL
PUBLIC, QUASI PUBLIC & OPEN SPACE
PUBLIC & QUASI PUBLIC
PARKS & RECREATION
OPEN SPACE PRESERVE
OPEN SPACE RECREATION
OPEN SPACE
PROPOSED FREEWAY COVER
WATER
SPECIAL PLAN AREA
RESORT
SCC
SPECIALTY CONFERENCE CENTER
VC
VILLAGE CORE
EASTERN URBAN CENTER
BAYFRONT PLANNING AREA
TRANSIT STATION
EXISTING
PROPOSED
JUNE 1, 2004

Scripps Mercy Hospital
Chula Vista
435 H Street
Chula Vista, CA 91910-4307
Tel 619-691-7000

Scripps

November 1, 2005

Mark Stephens
Principal Planner
City of Chula Vista
Department of Planning and Building
276 Fourth Avenue
Chula Vista, CA 91910

Dear Mr. Stephens:

J-1 Scripps Mercy Hospital Chula Vista is proud to continue our mission of providing quality and accessible health care to the residents of Chula Vista and the South County community. We also look forward to participating in the future redevelopment plans for Chula Vista's urban core.

We have reviewed the Revised Draft General Plan Update and Re-Circulated Draft Environmental Impact Report and are in support of the inclusion of the recommendation to conduct a special study to examine the potential for higher land use intensities and taller buildings along the H Street Transit focus corridor between Interstate 5 and Third Avenue into the General Plan. The results of this study will provide important information that will be necessary for Scripps' long range strategic planning efforts.

Sincerely,

Todd Hoff
Chief Operating Officer

RESPONSE

J-1 These comments do not reflect on the adequacy of the EIR. The comment, however, will be forwarded it to the appropriate City decision making body.

Environmental Health Coalition

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ehc@environmentalhealth.org ♦ www.environmentalhealth.org

November 2, 2005

Mr. Ed Batchelder
Planning Department
HAND DELIEVERED

RE: EHC comments on GPU Draft Environmental Impact Statement (DEIR)

Dear Mr. Batchelder:

Environmental Health Coalition (EHC) has participated in the General Plan Update process for several years. The new GPU is much improved and we thank you for the many revisions that the staff has included in the current edition. Our remaining concerns relate to analysis and mitigation of impacts to human health from known sources of air pollution. These should be addressed in the Air Quality section of the DEIR.

Request stronger language for avoiding human health risks from freeway pollution

K-1 We did not find any analysis or mention in the DEIR of a significant law passed in 2003 SB 352 Escutia which amended Section 17213 of the Education Code, and Section 21151.8 of the Public Resources Code, relating to public schools. This law disallows location of a school within 500 feet of a freeway due to health concerns except under very limited and site specific conditions. Also, the Air Resources Board Land Use Guidance is not referenced as a relevant guidance document that the City will comply with in its planning decisions. Both of these should be included in the EIR. Relative to adjacent location of highways and sensitive receptors we strongly believe that EE 6.10 should be revised to reflect current science and land use guidance related to sensitive uses and freeways in order to mitigate potential health impacts from polluted air. The current language implies that there are federal and state standards in existence to protect people from locating housing and schools from the impacts of highway pollution.

K-2 The Air Resources Board's *Land Use Planning Guidance* adopted this year that recommends a 500 foot buffer between freeways and sensitive receptors due to known health impacts in that zone. In effect, the ARB has already done a generic risk assessment and has decided it is a significant health risk to locate sensitive receptors in the 500 foot zone. Additional studies continue to confirm this and there have been several news articles on this issue of late. We recommend that a policy be revised to prohibit sensitive uses in this zone (at least housing and schools) unless there is some finding of extremely over-riding benefit such as a pressing need for this land use which is not possible to meet in a more healthful location. In this kind of case, then the requirement to do an HRA would be pursued and attempts to mitigate made.

K-1 This comment states that SB352 and Section 21151.8 of the Public Resources Code should be included in the EIR. Section 21151.8 was amended by SB352. This bill prohibited the approval by a school district of a school site within 500 feet from the edge of a freeway or other busy traffic corridor. For the project area, these are roadways carrying over 100,000 vehicles per day. The proposed General Plan Update does not plan for a school site within 500 feet of a roadway carrying more than 100,000 vehicles per day. The dEIR addresses the siting of new sensitive receivers within 500 feet of highways in Policy EE 6.10 on Page 406. Policy EE 6.10 states:

> The siting of new sensitive receivers within 500 feet of highways resulting from development or redevelopment projects shall require the preparation of a health risk assessment as part of the CEQA review of the project. Attendant health risks identified in the HRA shall be feasibly mitigated to the maximum extent practicable in accordance with CEQA, in order to help ensure that applicable federal and state standards are not exceeded.

K-2 This comment states that stronger language should be included for avoiding human health risks from freeway pollution and that a policy should be revised to prohibit sensitive uses within 500 feet of a freeway. This is a comment about the General Plan Update, rather than the dEIR. The dEIR addresses Policy EE 6.10 on Page 406 which states:

> The siting of new sensitive receivers within 500 feet of highways resulting from development or redevelopment projects shall require the preparation of a health risk assessment as part of the CEQA review of the project. Attendant health risks identified in the HRA shall be feasibly mitigated to the maximum extent practicable in accordance with CEQA, in order to help ensure that applicable federal and state standards are not exceeded.

Furthermore, Policy EE 6.4 (Page 406 of the dEIR) states:

> Avoid siting new or re-powered energy generation facilities, and other major toxic air emitters within 1,000 feet of a sensitive receiver, or the placement of a sensitive receiver within 1,000 feet of a major toxic emitter.

Based on these policies, the dEIR concluded that the impacts that could result from exposing sensitive receptors to substantial pollutant concentrations were self-mitigated. The policy comment will be forwarded to the appropriate City decision making body. The California Air Resources Board Air Quality and Land Use Handbook is "advisory and does not establish regulatory standards of any kind."

We suggest that EE 6.10 be revised to read:

EHC PROPOSED Revision (EE 6.10)
The siting of sensitive receptors within 500 feet of highways resulting from development or redevelopment projects shall be avoided. In the event that there is an extreme need or mitigating site specific circumstance, overriding considerations can be made to this policy in conjunction with preparation of a health risk assessment as part of the CEQA review of the project and attendant health risks identified in the HRA being mitigated to the maximum extent practicable and in compliance with SB352.

K-3 We request that the studies attached to this letter be included in the FEIR to demonstrate the associated risks and support more stringent prohibitions regarding locating housing and schools in this 500 foot buffer area.

Updated information on BFGoodrich Emissions Inventory should be included in the EIR

K-4 We repeat our concern that the impacts of the BFGoodrich and the South Bay Power Plant are not adequately analyzed or disclosed in this environmental assessment. As we raised in our comments on the first DEIR, just because the facilities are not located directly within the planning area they are directly upwind and directly impact the planning areas. In fact, there are residents currently living within 800 feet of BFGoodrich and a schools nearby and downwind of both emissions sources. The land use patterns in this area create a need for current and precise information about the emissions and health risks from Goodrich's operations which are not disclosed in the DEIR and should be.

Past health risk assessments and lack of current information on operations leave considerable uncertainty about the health hazard from Goodrich emissions at downwind locations. This facility's first HRA, in 1992, showed a 10 per million cancer risk isopleth that extended 20 kilometers offsite. The 1993 HRA showed a much reduced isopleth, but the health risks were still significant off-site. An updated emissions estimate was completed in 1991, but no new isopleth maps have been generated. Currently, Goodrich has the region's highest reported annual emissions of hexavalent chromium, with an annual emission rate estimated at 9.2 lbs. As operations at this facility have changed, accurate information should be provided in all EIRs for projects that can be impacted by these air emissions. This information will be imperative for communities and government to make informed decisions to both protect human health and preserve the economic benefits to the region from this facility.

Thank you for the opportunity to comment on this document.

Sincerely,

Laura Hunter, Director
Clean Bay Campaign

RESPONSE

K-3 This comment requests that the studies attached to the letter should be included in the Final EIR. The letter and attachments are included as part of the Final EIR.

K-4 This comment states that the updated information on BFGoodrich emissions inventory should be included in the EIR. Current emissions inventories from BFGoodrich are unavailable. The most recently available health risk assessment for the Goodrich facility, on file with the San Diego APCD is dated February 7, 1997 and was used as the basis for the analysis provided in the dEIR. The results of that analysis are provided on Table 5.11-11, page 405 of the dEIR. It states that the Maximum Lifetime Cancer Risk per million for the BF Goodrich/Rohr Industry plant is 7.7; lifetime Cancer Burden ‹0.1; Chronic THI‹0.1; and Acute THI‹0.1.

KEY STUDIES ON AIR POLLUTION AND HEALTH EFFECTS NEAR HIGH-TRAFFIC AREAS

Compiled by the Environmental Law and Policy Center and the Sierra Club

Air Pollution from Busy Roads Linked to Shorter Life Spans for Nearby Residents

Dutch researchers looked at the effects of long-term exposure to traffic-related air pollutants on 5,000 adults. They found that people who lived near a main road were almost twice as likely to die from heart or lung disease and 1.4 times as likely to die from any cause compared with those who lived in less-trafficked areas. Researchers say these results are similar to those seen in previous U.S. studies on the effects of long-term exposure to traffic-related air pollution. The authors say traffic emissions contain many pollutants that might be responsible for the health risks, such as ultrafine particles, diesel soot, and nitrogen oxides, which have been linked to cardiovascular and respiratory problems.

Hoek, Brunekreef, Goldbohn, Fischer, van den Brandt. (2002). Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. Lancet, 360 (9341): 1203-9.

Truck Traffic Linked to Childhood Asthma Hospitalizations

A study in Erie County, New York (excluding the city of Buffalo) found that children living in neighborhoods with heavy truck traffic within 200 meters of their homes had increased risks of asthma hospitalization. The study examined hospital admission for asthma amongst children ages 0-14, and residential proximity to roads with heavy traffic.

Lin, Munsie, Hwang, Fitzgerald, and Cayo. (2002). Childhood Asthma Hospitalization and Residential Exposure to State Route Traffic. Environmental Research, Section A, Vol. 88, pp. 73-81.

Pregnant Women Who Live Near High Traffic Areas More Likely to Have Premature and Low Birth Weight Babies.

Researchers observed an approximately 10-20% increase in the risk of premature birth and low birth weight for infants born to women living near high traffic areas in Los Angeles County. In particular, the researchers found that for each one part per million increase in annual average carbon monoxide concentrations where the women lived, there was a 19% and 11 % increase in risk for low birth weight and premature births, respectively.

Wilhelm, Ritz. (2002). Residential Proximity to Traffic and Adverse Birth Outcomes in Los Angeles County, California, 1994-1996. Environmental Health Perspectives. doi: 10.1289/ehp.5688.

Traffic-Related Air Pollution Associated with Respiratory Symptoms in Two Year Old Children.

This cohort study found that two year old children who are exposed to higher levels of traffic-related air pollution are more likely to have self-reported respiratory illnesses, including wheezing, ear/nose/throat infections, and reporting of physician-diagnosed asthma, flu or serious cold.

Brauer et al. (2002). Air Pollution from Traffic and the Development of Respiratory Infections and Asthmatic and Allergic Symptoms in Children. Am J Respiratory and Critical Care Medicine. Vol. 166 pp 1092-1098.

People Who Live Near Freeways Exposed to 25 Times More Particle Pollution

Studies conducted in the vicinity of Interstates 405 and 710 in southern California found that the number of ultrafine particles in the air was approximately 25 times more concentrated near the freeways and that pollution levels gradually decrease to near normal (background) levels around 300 meters, or 990 feet, downwind from the freeway. The researchers note that motor vehicles are the most significant source of ultrafine particles, which have been linked to increases in mortality and morbidity. Recent research concludes that ultrafine particles are more toxic than larger particles with the same chemical composition. Moreover, the researchers found considerably higher concentrations of carbon monoxide pollution near the freeways.

Zhu, Hinds, Kim, Sioutas. Concentration and size distribution of ultrafine particles near a major highway. Journal of the Air and Waste Management Association.. September 2002.

Zhu, Hinds, Kim, Shen, Sioutas. Study of ultrafine particles near a major highway with heavy-duty diesel traffic. Atmospheric Environment. 36(2002),4323-4335.

Asthma More Common for Children Living Near Freeways.

A study of nearly 10,000 children in England found that wheezing illness, including asthma, was more likely with increasing proximity of a child's home to main roads. The risk was greatest for children living within 90 meters of the road.

Venn et al. (2001). Living Near A Main Road and the Risk of Wheezing Illness in Children. American Journal of Respiratory and Critical Care Medicine. Vol. 164, pp 2177-2180.

A study of 1,068 Dutch children found that asthma, wheeze, cough, and runny nose were significantly more common in children living within 100 meters of freeways. Increasing density of truck traffic was also associated with significantly higher asthma levels - particularly in girls.

van Vliet et al. (1997). Motor exhaust and chronic respiratory symptoms in children living near freeways. Environmental Research. 74:12-132.

Children Living Near Busy Roads More Likely to Develop Cancer

A 2000 Denver study showed that children living within 250 yards of streets or highways with 20,000 vehicles per day are six times more likely to develop all types of cancer and eight times

more likely to get leukemia. The study looked at associations between traffic density, power lines, and all childhood cancers with measurements obtained in 1979 and 1990. It found a weak association from power lines, but a strong association with highways. It suggested that benzene pollution might be the cancer promoter causing the problem.

Pearson et al. (2000). Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers. Journal of Air and Waste Management Association 50:175-180.

Most Traffic-Related Deaths Due to Air Pollution, Not Traffic Accidents

Another study analyzed the affect of traffic-related air pollution and traffic accidents on life expectancy in the area of Baden-Wurttemberg, Germany. It estimated that 4325 deaths in this region would result from motor vehicle emissions compared to 891 from traffic accidents (over a lifetime).

Szagun and Seidel. (2000). Mortality due to road traffic in Baden-Aurttemberg - air pollution, accidents, noise. Gesundheitswesen. 62(4): 225-33.

Emissions from Motor Vehicles Dominate Cancer Risk

The most comprehensive study of urban toxic air pollution ever undertaken shows that motor vehicles and other mobile sources of air pollution are the predominant source of cancer-causing air pollutants in Southern California. Overall, the study showed that motor vehicles and other mobile sources accounted for about 90% of the cancer risk from toxic air pollution, most of which is from diesel soot (70% of the cancer risk). Industries and other stationary sources accounted for the remaining 10%. The study showed that the highest risk is in urban areas where there is heavy traffic and high concentrations of population and industry.

South Coast Air Quality Management District. Multiple Air Toxics Exposure Study-II. March 2000.

Cancer Risk Higher Near Major Sources of Air Pollution, Including Highways

A 1997 English study found a cancer corridor within three miles of highways, airports, power plants, and other major polluters. The study examined children who died of leukemia or other cancers from the years 1953-1980, where they were born and where they died. It found that the greatest danger lies a few hundred yards from the highway or pollution facility and decreases as you get away from the facility.

Knox and Gilman (1997). Hazard proximities of childhood cancers in Great Britain from 1953-1980. Journal of Epidemiology and Community Health. 51: 151-159.

A School's Proximity to Freeways Associated with Asthma Prevalence

A study of 1498 children in 13 schools in the Province of South Holland found a positive relationship between school proximity to freeways and asthma occurrence. Truck traffic intensity and the concentration of emissions measured in schools were found to be significantly associated with chronic respiratory symptoms.

Speizer, F. E. and B. G. Ferris, Jr. (1973). Exposure to automobile exhaust. I. Prevalence of respiratory symptoms and disease. Archives of Environmental Health. 26(6): 313-8. van Vliet, P., M. Knape, et al. (1997). Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. Environmental Research.. 74(2): 122-32.

Lung Function Reduction Among Children More Likely if Living Near Truck Traffic

A European study determined that exposure to traffic-related air pollution, `in particular diesel exhaust particles,' may lead to reduced lung function in children living near major motorways.

Brunekreef B; Janssen NA; de Hartog J; Harssema H; Knape M; van Vliet P. (1997). "Air pollution from truck traffic and lung function in children living near motorways." Epidemiology. 8(3):298-303.

Asthma Symptoms Caused by Truck Exhaust

A study was conducted in Munster, Germany to determine the relationship between truck traffic and asthma symptoms. In total, 3,703 German students, between the ages of 12-15 years, completed a written and video questionnaire in 1994-1995. Positive associations between both wheezing and allergic rhinitis and truck traffic were found during a 12-month period. Potentially confounding variables, including indicators of socio-economic status, smoking, etc., did not alter the associations substantially.

Duhme, H., S. K. Weiland, et al. (1996). The association between self-reported symptoms of asthma and allergic rhinitis and self-reported traffic density on street of residence in adolescents. Epidemiology7(6): 578-82.

Proximity of a Child's Residence to Major Roads Linked to Hospital Admissions for Asthma

A study in Birmingham, United Kingdom, determined that living near major roads was associated with the risk of hospital admission for asthma in children younger than 5 yrs of age. The area of residence and traffic flow patterns were compared for children admitted to the hospital for asthma, children admitted for nonrespiratory reasons, and a random sample of children from the community. Children admitted with an asthma diagnosis were significantly more likely to live in an area with high traffic flow (> 24,000 vehicles/ 24 hrs) located along the nearest segment of main road than were children admitted for nonrespiratory reasons or children form the community.

Edwards, J., S. Walters, et al. (1994). Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. Archives of Environmental Health. 49(4): 223-7.

Exposure to Carcinogenic Benzene Higher for Children Living Near High Traffic Areas

German researchers compared forty-eight children who lived in a central urban area with high traffic density with seventy-two children who lived in a small city with low traffic density. They found that the blood levels of benzene in children who lived in the high-traffic-density area were

71% higher than those of children who lived in the low-traffic-density area. Blood levels of toluene and carboxyhemoglobin (formed after breathing carbon monoxide) were also significantly elevated (56% and 33% higher, respectively) among children regularly exposed to vehicle emissions. Aplastic anemia and leukemia are associated with excessive exposure to benzene.

Jermann E, Hajimiragha H, Brockhaus A, Freier I, Ewers U, Roscovanu A: Exposure of children to benzene and other motor vehicle emissions. Zentralblatt fur Hygiene and Umweltmedizin 189:50-61, 1989.

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The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age

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ABSTRACT

BACKGROUND

Whether exposure to air pollution adversely affects the growth of lung function during the period of rapid lung development that occurs between the ages of 10 and 18 years is unknown.

METHODS

In this prospective study, we recruited 1759 children (average age, 10 years) from schools in 12 southern California communities and measured lung function annually for eight years. The rate of attrition was approximately 10 percent per year. The communities represented a wide range of ambient exposures to ozone, acid vapor, nitrogen dioxide, and particulate matter. Linear regression was used to examine the relationship of air pollution to the forced expiratory volume in one second (FEV_1) and other spirometric measures.

RESULTS

Over the eight-year period, deficits in the growth of FEV_1 were associated with exposure to nitrogen dioxide (P=0.005), acid vapor (P=0.004), particulate matter with an aerodynamic diameter of less than 2.5 μm ($PM_{2.5}$) (P=0.04), and elemental carbon (P=0.007), even after adjustment for several potential confounders and effect modifiers. Associations were also observed for other spirometric measures. Exposure to pollutants was associated with clinically and statistically significant deficits in the FEV_1 attained at the age of 18 years. For example, the estimated proportion of 18-year-old subjects with a low FEV_1 (defined as a ratio of observed to expected FEV_1 of less than 80 percent) was 4.9 times as great at the highest level of exposure to $PM_{2.5}$ as at the lowest level of exposure (7.9 percent vs. 1.6 percent, P=0.002).

CONCLUSIONS

The results of this study indicate that current levels of air pollution have chronic, adverse effects on lung development in children from the age of 10 to 18 years, leading to clinically significant deficits in attained FEV_1 as children reach adulthood.

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PR-65

THERE IS MOUNTING EVIDENCE THAT air pollution has chronic, adverse effects on pulmonary development in children. Longitudinal studies conducted in Europe[1-3] and the United States[4-6] have demonstrated that exposure to air pollution is associated with reductions in the growth of lung function, strengthening earlier evidence[7-12] based on cross-sectional data. However, previous longitudinal studies have followed young children for relatively short periods (two to four years), leaving unresolved the question of whether the effects of air pollution persist from adolescence into adulthood. The Children's Health Study[13] enrolled children from 12 southern California communities representing a wide range of exposures to ambient air pollution. We documented the children's respiratory growth from the ages of 10 to 18 years. Over this eight-year period, children have substantial increases in lung function. By the age of 18 years, girls' lungs have nearly matured, and the growth in lung function in boys has slowed considerably, as compared with the rate in earlier adolescence.[14] We analyzed the association between long-term exposure to ambient air pollution and the growth in lung function over the eight-year period from the ages of 10 to 18 years. We also examined whether any observed effect of air pollution on this eight-year growth period results in clinically significant deficits in attained lung function at the age of 18 years.

METHODS

STUDY SUBJECTS

In 1993, the Children's Health Study recruited 1759 fourth-grade children (average age, 10 years) from elementary schools in 12 southern California communities as part of an investigation of the long-term effects of air pollution on children's respiratory health.[6,12,13] Data on pulmonary function were obtained by trained field technicians, who traveled to study schools annually from the spring of 1993 through the spring of 2001 to perform maximal-effort spirometric testing of the children. Details of the testing protocol have been published previously.[12] We analyzed three measures of pulmonary function: forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1), and maximal midexpiratory flow rate (MMEF). Pulmonary-function tests were not performed on any child who was absent from school on the day of testing, but such a child was still eligible for testing in subsequent years. Children who moved away from their recruitment community were classified as lost to follow-up and were not tested further. From the initial sample of the 1759 children in 1993, the number of children available for follow-up was 1414 in 1995, 1252 in 1997, 1031 in 1999, and 747 in 2001, reflecting the attrition of approximately 10 percent of subjects per year.

A baseline questionnaire, completed at study entry by each child's parents or legal guardian, was used to obtain information on the children's characteristics, including race, presence or absence of Hispanic ethnic background, level of parental education, presence or absence of a history of asthma diagnosed by a doctor, exposure to maternal smoking in utero, and household exposure to gas stoves, pets, and environmental tobacco smoke. Questions administered at the time of annual pulmonary-function testing were used to update information on asthma status, personal smoking status, and exposure to environmental tobacco smoke. The distribution of baseline characteristics of all study subjects and of two subgroups defined according to the length of follow-up (all eight years or less than eight years) is shown in Supplementary Appendix 1 (available with the full text of this article at www.nejm.org). The length of follow-up was significantly associated with factors related to the mobility of the population, including race, presence or absence of Hispanic ethnic background, presence or absence of exposure to environmental tobacco smoke, and parents' level of education. However, the length of follow-up was not significantly associated with baseline lung function or the level of exposure to air pollution, suggesting that the loss to follow-up did not differ with respect to the primary variables of interest.

The study protocol was approved by the institutional review board for human studies at the University of Southern California, and written informed consent was provided by a parent or legal guardian for all study subjects. We did not obtain assent from minor children, since this was not standard practice when the study was initiated.

AIR-POLLUTION DATA

Air-pollution–monitoring stations were established in each of the 12 study communities and provided continuous data, beginning in 1994. Each station measured average hourly levels of ozone, nitrogen

dioxide, and particulate matter with an aerodynamic diameter of less than 10 μm (PM_{10}). Stations also collected two-week integrated-filter samples for measuring acid vapor and the mass and chemical makeup of particulate matter with an aerodynamic diameter of less than 2.5 μm ($PM_{2.5}$). Acid vapor included both inorganic acids (nitric and hydrochloric) and organic acids (formic and acetic). For statistical analysis, we used total acid, computed as the sum of nitric, formic, and acetic acid levels. Hydrochloric acid was excluded from this sum, since levels were very low and close to the limit of detection. In addition to measuring $PM_{2.5}$, we determined the levels of elemental carbon and organic carbon, using method 5040 of the National Institute for Occupational Safety and Health.[15] We computed annual averages on the basis of average levels in a 24-hour period in the case of PM_{10} and nitrogen dioxide, and a two-week period in the case of $PM_{2.5}$, elemental carbon, organic carbon, and acid vapor. For ozone, we computed the annual average of the levels obtained from 10 a.m. to 6 p.m. (the eight-hour daytime average) and of the one-hour maximal levels. We also calculated long-term mean pollutant levels (from 1994 through 2000) for use in the statistical analysis of the lung-function outcomes.

STATISTICAL ANALYSIS

The outcome data consisted of the results of 5454 pulmonary-function tests of 876 girls and 5300 tests of 883 boys over the eight-year period. We adopted a two-stage regression approach to relate the longitudinal pulmonary-function data for each child to the average air-pollution levels in each study community.

The first-stage model was a regression of each pulmonary-function measure (values were log-transformed) on age to obtain separate, community-specific average growth curves for girls and boys. To account for the growth pattern during this period, we used a linear spline model[14] that consisted of four straight lines over the age intervals of younger than 12 years, 12 to 14 years, 14 to 16 years, and older than 16 years, constrained to be connected at the three "knot" points. The model included adjustments for log values for height; body-mass index (the weight in kilograms divided by the square of the height in meters); the square of the body-mass index; race; the presence or absence of Hispanic ethnic background, doctor-diagnosed asthma, any tobacco smoking by the child in the preceding year, exposure to environmental tobacco smoke, and exercise or respiratory tract illness on the day of the test; and indicator variables for the field technician and the spirometer. In addition to these covariates, random effects were included to account for the multiple measurements contributed by each subject. An analysis of residual values confirmed that the assumptions of the model had been satisfied. The first-stage model was used to estimate the mean and variance of the growth in lung function over the eight-year period in each of the 12 communities, separately for girls and boys.

The second-stage model was a linear regression of the 24 sex- and community-specific estimates of the growth in lung function over the eight-year period on the corresponding average levels of each air pollutant in each community. Inverses of the first-stage variances were incorporated as weights, and a community-specific random effect was included to account for residual variation between communities. A sex-by-pollutant interaction was included in the model to evaluate whether there was a difference in the effect of a given pollutant between the sexes, and when this value was nonsignificant, the model was refitted to estimate the sex-averaged effect of the pollutant. Pollutant effects are reported as the difference in the growth in lung function over the eight-year period from the least to the most polluted community, with negative differences indicative of growth deficits with increasing exposure. We also considered two-pollutant models obtained by simultaneously regressing the growth in lung function over the eight-year period on pairs of pollutants.

In addition to examining the growth in lung function over the eight-year period, we analyzed the FEV_1 measurements obtained in 746 subjects during the last year of follow-up (average age, 17.9 years) to determine whether exposure to air pollution was associated with clinically significant deficits in attained FEV_1. We defined a low FEV_1 as an attained FEV_1 below 80 percent of the predicted value, a criterion commonly used in clinical settings to identify persons who are at increased risk for adverse respiratory conditions. To determine the predicted FEV_1, we first fitted a regression model for observed FEV_1 (using log-transformed values) with the following predictors: log-transformed height, body-mass index, the square of the body-mass index, sex, race or ethnic group, asthma status, field technician, and interactions between sex and log-transformed height, sex and asthma, and sex and

race or ethnic group. This model explained 71 percent of the variance in the attained FEV_1 level. For each subject, we then computed the predicted FEV_1 from the model and considered subjects to have a low FEV_1 if the ratio of observed to predicted FEV_1 was less than 80 percent. Linear regression was then used to examine the correlation between the community-specific proportion of subjects with a low FEV_1 and the average level of each pollutant from 1994 through 2000. This model included a community-specific random effect to account for residual variation. Regression procedures in SAS software[16] were used to fit all models. Associations denoted as statistically significant were those that yielded a P value of less than 0.05, assuming a two-sided alternative hypothesis.

RESULTS

From 1994 through 2000, there was substantial variation in the average levels of study pollutants across the 12 communities, with relatively little year-to-year variation in the annual levels within each community (Fig. 1). From 1994 through 2000, the

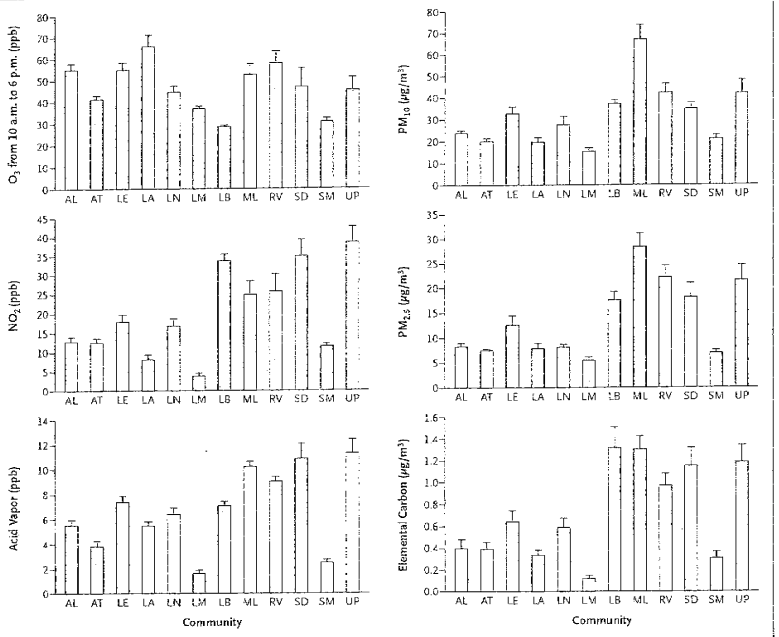

Figure 1. **Mean (+SD) Annual Average Levels of Pollutants from 1994 through 2000 in the 12 Study Communities in Southern California.**
AL denotes Alpine, AT Atascadero, LE Lake Elsinore, LA Lake Arrowhead, LN Lancaster, LM Lompoc, LB Long Beach, ML Mira Loma, RV Riverside, SD San Dimas, SM Santa Maria, and UP Upland. O_3 denotes ozone, NO_2 nitrogen dioxide, and PM_{10} and $PM_{2.5}$ particulate matter with an aerodynamic diameter of less than 10 μm and less than 2.5 μm, respectively.

average levels of ozone were not significantly correlated across communities with any other study pollutant (Table 1). However, correlations between other pairs of pollutants were all significant, ranging from an R of 0.64 ($P<0.05$) for nitrogen dioxide and organic carbon, to an R of 0.97 ($P<0.001$) for PM_{10} and organic carbon. Thus, nitrogen dioxide, acid vapor, and the particulate-matter pollutants can be regarded as a correlated "package" of pollutants with a similar pattern relative to each other across the 12 communities.

Among the girls, the average FEV_1 increased from 1988 ml at the age of 10 years to 3332 ml at the age of 18 years, yielding an average growth in FEV_1 of 1344 ml over the eight-year period (Table 2). The corresponding averages in boys were 2082 ml and 4464 ml, yielding an average growth in FEV_1 of 2382 ml over the eight-year period. Similar patterns of growth over the eight-year period were observed for FVC and MMEF (Table 2).

Although the average growth in FEV_1 was larger in boys than in girls, the correlations of growth with air pollution did not differ significantly between the sexes, as shown for nitrogen dioxide in Figure 2. The sex-averaged analysis, depicted by the regression line in Figure 2, demonstrated a significant negative correlation between the growth in FEV_1 over the eight-year period and the average nitrogen dioxide level ($P=0.005$). The estimated difference in the average growth in FEV_1 over the eight-year period from the community with the lowest nitrogen dioxide level to the community with the highest nitrogen dioxide level, represented by the slope of the plotted regression line in Figure 2, was −101.4 ml.

Estimated differences in the growth of FEV_1, FVC, and MMEF during the eight-year period with respect to all pollutants are summarized in Table 3. Deficits in the growth of FEV_1 and FVC were observed for all pollutants, and deficits in the growth of MMEF were observed for all but ozone, with several combinations of outcome variables and pollutants attaining statistical significance. Specifically, for FEV_1 we observed significant negative correlations between the growth in this variable over the eight-year period and exposure to acid vapor ($P=0.004$), $PM_{2.5}$ ($P=0.04$), and elemental carbon ($P=0.007$), in addition to the above-mentioned correlation with nitrogen dioxide. As with FEV_1, the effects of the various pollutants on FVC and MMEF did not differ significantly between boys and girls. Significant deficits in FVC were associated with exposure to nitrogen dioxide ($P=0.05$) and acid vapor ($P=0.03$), whereas deficits in MMEF were associated with exposure to nitrogen dioxide ($P=0.02$) and elemental carbon ($P=0.04$). There was no significant evidence that ozone, either the average value obtained from 10 a.m. to 6 p.m. or the one-hour maximal level, was associated with any measure of lung function. In two-pollutant models for any of the measures of pulmonary function, adjustment for ozone did not substantially alter the effect estimates or significance levels of any other pollutant (data not shown). In general, two-pollutant models for any pair of pollutants did not provide a significantly better fit to the data than the corre-

Table 1. **Correlation of Mean Air-Pollution Levels from 1994 through 2000 across the 12 Study Communities.***

Pollutant	O_3 (10 a.m.–6 p.m.)	NO_2	Acid Vapor†	PM_{10}	$PM_{2.5}$	Elemental Carbon	Organic Carbon
				R value			
O_3							
1 Hr max	0.98	0.10	0.53	0.31	0.33	0.17	0.25
10 a.m.–6 p.m.		−0.11	0.35	0.18	0.18	−0.03	0.13
NO_2			0.87	0.67	0.79	0.94	0.64
Acid vapor†				0.79	0.87	0.88	0.76
PM_{10}					0.95	0.85	0.97
$PM_{2.5}$						0.91	0.91
Elemental carbon							0.82

* Unless otherwise noted, values are the 24-hour average pollution levels. O_3 denotes ozone, NO_2 nitrogen dioxide, and PM_{10} and $PM_{2.5}$ particulate matter with an aerodynamic diameter of less than 10 μm and less than 2.5 μm, respectively.
† Acid vapor is the sum of nitric, formic, and acetic acid levels.

TABLE 2. Mean Levels of Growth in Pulmonary Function during the Eight-Year Study Period, from 1993 to 2001.*

Pulmonary-Function Measure	Girls			Boys		
	Age of 10 yr	Age of 18 yr	Average 8-yr growth	Age of 10 yr	Age of 18 yr	Average 8-yr growth
FVC (ml)	2262	3790	1528	2427	5202	2775
FEV_1 (ml)	1988	3332	1344	2082	4464	2382
MMEF (ml/sec)	2311	3739	1428	2287	4709	2422

* Levels at the ages of 10 and 18 years are derived from the growth model described in the Methods section. FVC denotes forced vital capacity, FEV_1 forced expiratory volume in one second, and MMEF maximal midexpiratory flow rate.

sponding single-pollutant models; this was not surprising, given the strong correlation between most pollutants.

The association between pollution and the growth in FEV_1 over the eight-year period remained significant in a variety of sensitivity analyses (Table 4). For example, estimates of the effect of acid vapor and elemental carbon (model 1 in Table 4) changed little with adjustment for in-utero exposure to maternal smoking (model 2), presence in the home of a gas stove (model 3) or pets (model 4), or parental level of education (model 5). To account for possible confounding by short-term effects of air pollution, we fitted a model that adjusted for the average ozone, nitrogen dioxide, and PM_{10} levels on the three days before each child's pulmonary-function test. This adjustment also had little effect on the estimates of the long-term effects of air pollution (model 6). Table 4 also shows that the effects of pollutants remained large and significant in the subgroups of children with no history of asthma (model 7) and those with no history of smoking (model 8). The effects of pollutants were not significant among the 457 children who had a history of asthma or among the 483 children who had ever smoked (data not shown), although the sample sizes in these subgroups were small. Model 9 demonstrates that the extremes in pollutant levels did not drive the observed associations; in other words, we found similar effect estimates after eliminating the two communities with the highest and lowest levels of each pollutant. Finally, model 10 shows the effects of pollutants in the subgroup of subjects who underwent pulmonary-function testing in both 1993 and 2001 (i.e., subjects who participated in both the first and last year of the study). The magnitudes of effects in this subgroup were similar to those in the entire sample (Model 1), suggesting that observed effects of pollutants in the entire sample cannot be attributed to biased losses to follow-up across communities. These sensitivity analyses were also applied to the other pollutants and to FVC and MMEF, with similar results.

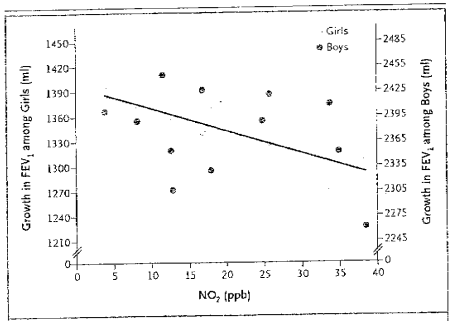

Figure 2. Community-Specific Average Growth in FEV_1 among Girls and Boys During the Eight-Year Period from 1993 to 2001 Plotted against Average Nitrogen Dioxide (NO_2) Levels from 1994 through 2000.

Pollution-related deficits in the average growth in lung function over the eight-year period resulted in clinically important deficits in attained lung function at the age of 18 years (Fig. 3). Across the 12 communities, a clinically low FEV_1 was positively correlated with the level of exposure to nitrogen dioxide ($P=0.005$), acid vapor ($P=0.01$), PM_{10} ($P=0.02$), $PM_{2.5}$ ($P=0.002$), and elemental carbon ($P=0.006$). For example, the estimated proportion of children with a low FEV_1 (represented by the regression line in Fig. 3) was 1.6 percent at the lowest level of exposure to $PM_{2.5}$ and was 4.9 times as great (7.9 percent) at the highest level of exposure to $PM_{2.5}$

Table 3. Difference in Average Growth in Lung Function over the Eight-Year Study Period from the Least to the Most Polluted Community.*

Pollutant	FVC		FEV_1		MMEF	
	Difference (95% CI)	P Value	Difference (95% CI)	P Value	Difference (95% CI)	P Value
	ml		*ml*		*ml/sec*	
O_3						
10 a.m.–6 p.m.	−50.6 (−171.0 to 69.7)	0.37	−22.8 (−122.3 to 76.6)	0.62	85.6 (−130.0 to 301.1)	0.40
1-Hr max	−70.3 (−183.3 to 42.6)	0.20	−44.5 (−138.9 to 50.0)	0.32	45.7 (−172.3 to 263.6)	0.65
NO_2	−95.0 (−189.4 to −0.6)	0.05	−101.4 (−164.5 to −38.4)	0.005	−211.0 (−377.6 to −44.4)	0.02
Acid vapor	−105.2 (−194.5 to −15.9)	0.03	−105.8 (−168.8 to −42.7)	0.004	−165.0 (−344.8 to 14.7)	0.07
PM_{10}	−60.2 (−190.6 to 70.3)	0.33	−82.1 (−176.9 to 12.8)	0.08	−154.2 (−378.3 to 69.8)	0.16
$PM_{2.5}$	−60.1 (−166.1 to 45.9)	0.24	−79.7 (−153.0 to −6.4)	0.04	−168.9 (−345.5 to 7.8)	0.06
Elemental carbon	−77.7 (−166.7 to 11.3)	0.08	−87.9 (−146.4 to −29.4)	0.007	−165.5 (−323.4 to −7.6)	0.04
Organic carbon	−58.6 (−196.1 to 78.8)	0.37	−86.2 (−185.6 to 13.3)	0.08	−151.2 (−389.4 to 87.1)	0.19

* Values are the differences in the estimated rate of eight-year growth at the lowest and highest observed levels of the indicated pollutant. Differences are scaled to the range across the 12 study communities in the average level of each pollutant from 1994 through 2000 as follows: 37.5 ppb of O_3 (measured from 10 a.m. to 6 p.m.), 46.0 ppb of O_3 (the one-hour maximal level), 34.6 ppb of NO_2, 9.6 ppb of acid vapor, 51.4 μg of PM_{10} per cubic meter, 22.8 μg of $PM_{2.5}$ per cubic meter, 1.2 μg of elemental carbon per cubic meter, and 10.5 μg of organic carbon per cubic meter. CI denotes confidence interval.

(P=0.002). Similar associations between these pollutants and a low FEV_1 were observed in the subgroup of children with no history of asthma and the subgroup with no history of smoking (data not shown). A low FEV_1 was not significantly correlated with exposure to ozone in any group.

DISCUSSION

The results of this study provide robust evidence that lung development, as measured by the growth in FVC, FEV_1, and MMEF from the ages of 10 to 18 years, is reduced in children exposed to higher levels of ambient air pollution. The strongest associations were observed between FEV_1 and a correlated set of pollutants, specifically nitrogen dioxide, acid vapor, and elemental carbon. The effects of these pollutants on FEV_1 were similar in boys and girls and remained significant among children with no history of asthma and among those with no history of smoking, suggesting that most children are susceptible to the chronic respiratory effects of breathing polluted air. The magnitude of the observed effects of air pollution on the growth in lung function during this age interval was similar to those that have been reported for exposure to maternal smoking[17,18] and smaller than those reported for the effects of personal smoking.[17,19]

Cumulative deficits in the growth in lung function during the eight-year study period resulted in a strong association between exposure to air pollution and a clinically low FEV_1 at the age of 18 years. In general, lung development is essentially complete in girls by the age of 18 years, whereas in boys it continues into their early 20s, but at a much reduced rate. It is therefore unlikely that clinically significant deficits in lung function at the age of 18 years will be reversed in either girls or boys as they make the transition into adulthood. Deficits in lung function during young adulthood may increase the risk of respiratory conditions — for example, episodic wheezing that occurs during a viral infection.[20] However, the greatest effect of pollution-related deficits may occur later in life, since reduced lung function is a strong risk factor for complications and death during adulthood.[21-27]

Deficits in lung function were associated with a correlated set of pollutants that included nitrogen dioxide, acid vapor, fine-particulate matter ($PM_{2.5}$), and elemental carbon. In southern California, the primary source of these pollutants is motor vehicles, either through direct tailpipe emissions or downwind physical and photochemical reactions of vehicular emissions. Both gasoline- and diesel-powered engines contribute to the tons of pollutants exhausted into southern California's air every day, with diesel vehicles responsible for disproportionate amounts of nitrogen dioxide, $PM_{2.5}$, and ele-

Table 4. **Sensitivity Analysis of the Effects of Acid Vapor and Elemental Carbon on Growth in FEV_1 over the Eight-Year Study Period.***

Model	Acid Vapor	Elemental Carbon
	Difference (95% Confidence Interval)	
Main model (model 1)†	−105.8 (−168.8 to −42.7)	−87.9 (−146.4 to −29.4)
Additional covariates‡		
Main model + in-utero exposure to maternal smoking (model 2)	−108.8 (−173.3 to −44.2)	−85.8 (−147.4 to −24.1)
Main model + exposure to gas stove (model 3)	−106.0 (−181.5 to −30.6)	−84.8 (−154.7 to −14.9)
Main model + pets in home (model 4)	−108.4 (−171.6 to −45.2)	−89.8 (−149.1 to −30.6)
Main model + parental level of education (model 5)	−100.7 (−167.2 to −34.2)	−80.9 (−142.7 to −19.0)
Main model + short-term effects of pollution (model 6)§	−112.4 (−201.4 to −23.3)	−103.2 (−181.8 to −24.5)
Subgroup effects		
No history of asthma (model 7)¶	−98.1 (−166.4 to −29.8)	−88.9 (−149.2 to −28.6)
No history of smoking (model 8)‖	−115.6 (−233.7 to 2.5)	−113.3 (−214.9 to −11.6)
After exclusion of communities with lowest and highest levels of pollution (model 9)**	−106.7 (−192.3 to −21.2)	−94.7 (−173.7 to −15.7)
Complete follow-up (model 10)††	−132.4 (−226.2 to −38.7)	−97.4 (−195.6 to 0.9)

* Values are the differences in the estimated rate of eight-year growth at the lowest and highest observed levels of the indicated pollutant. Differences are scaled to the range across the 12 study communities in the average level of each pollutant from 1994 through 2000 as follows: 9.6 ppb of acid vapor and 1.2 μg of elemental carbon per cubic meter.

† Model 1 is equivalent to effect estimates for FEV_1 in Table 3 and is based on data on 1759 children.

‡ The main model was adjusted for each of the covariates listed.

§ Values were adjusted for the average levels of O_3, NO_2, and PM_{10} on the three days before each child's pulmonary-function test.

¶ The analysis includes data on 1302 children with no history of doctor-diagnosed asthma.

‖ The analysis includes data on 1276 children with no history of active tobacco smoking at any time during follow-up.

** The analysis excludes children from the two communities with the lowest and highest levels of each pollutant. This leaves 1507 children (excluding those from Lompoc and Upland) in the analysis of acid vapor and 1484 children (excluding those from Lompoc and Long Beach) in the analysis of elemental carbon.

†† The analysis includes 713 children who underwent pulmonary-function testing in both 1993 and 2001 (i.e., those observed throughout the study).

mental carbon. In the current study, however, we could not discern the independent effects of pollutants because they came from common sources and there was a high degree of intercorrelation among them; similar difficulties have also been encountered in other studies of lung function and air-pollutant mixtures.[1,2,9,28-30] Since ozone is also formed during photochemical reactions involving fuel-combustion products, one might expect ozone to be correlated with the other study pollutants and therefore to show similar associations with lung function. However, the Children's Health Study was specifically designed to minimize the correlation of ozone with other pollutants across the 12 study communities. Thus, although ozone has been convincingly linked to acute health effects in many other studies,[11] our results provide little evidence that ambient ozone at current levels is associated with chronic deficits in the growth of lung function in children. Only a few other studies have addressed the long-term effects of ozone on lung development in children, and results have been inconsistent.[31] Although we found little evidence of an effect of ozone, this result needs to be interpreted with caution given the potential for substantial misclassification of exposure to ozone.[32,33]

The mechanism whereby exposure to pollutants could lead to reduced lung development is unknown, but there are many possibilities. Our observation of associations between air pollution and all three measures of lung function — FVC, FEV_1, and MMEF — suggests that more than one process is involved. FVC is largely a function of the number and size of alveoli, with differences in volume pri-

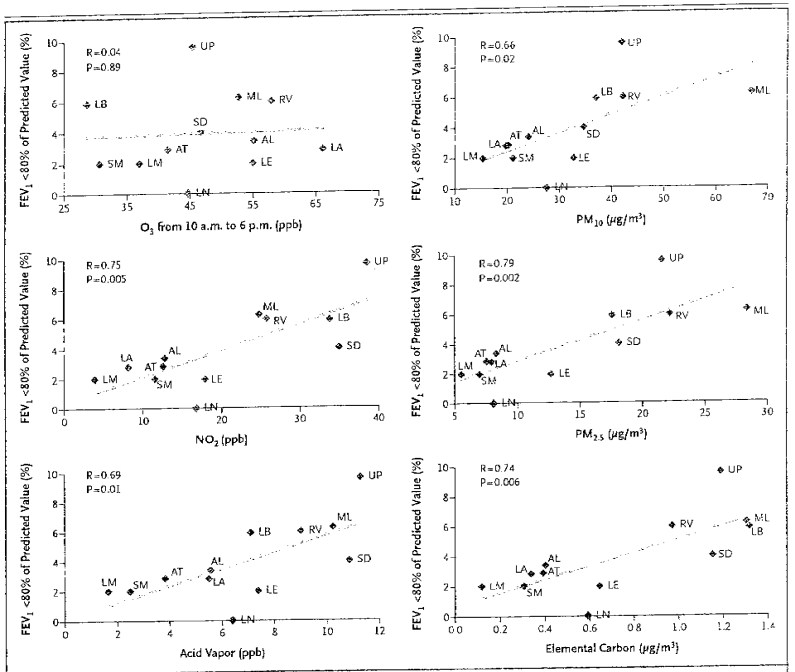

Figure 3. **Community-Specific Proportion of 18-Year-Olds with a FEV_1 below 80 Percent of the Predicted Value Plotted against the Average Levels of Pollutants from 1994 through 2000.**

The correlation coefficient (R) and P value are shown for each comparison. AL denotes Alpine, AT Atascadero, LE Lake Elsinore, LA Lake Arrowhead, LN Lancaster, LM Lompoc, LB Long Beach, ML Mira Loma, RV Riverside, SD San Dimas, SM Santa Maria, and UP Upland. O_3 denotes ozone, NO_2 nitrogen dioxide, and PM_{10} and $PM_{2.5}$ particulate matter with an aerodynamic diameter of less than 10 μm and less than 2.5 μm, respectively.

marily attributable to differences in the number of alveoli, since their size is relatively constant.[34] However, since the postnatal increase in the number of alveoli is complete by the age of 10 years, pollution-related deficits in the growth of FVC and FEV_1 during adolescence may, in part, reflect a reduction in the growth of alveoli. Another plausible mechanism of the effect of air pollution on lung development is airway inflammation, such as occurs in bronchiolitis; such changes have been observed in the airways of smokers and of subjects who lived in polluted environments.[35,36]

A strength of our study was the long-term, prospective follow-up of a large cohort, with exposure and outcome data collected in a consistent manner throughout the study period. As in any epidemiologic study, however, the observed effects could be biased by underlying associations of the exposure and outcome to some confounding variables. We adjusted for known potential confounders, includ-

ing personal characteristics and other sources of exposure to pollutants, but the possibility of confounding by other factors still exists. Over the eight-year follow-up period, approximately 10 percent of study subjects were lost to follow-up each year. Attrition is a potential source of bias in a cohort study if loss to follow-up is related to both exposure and outcome. However, we did not see evidence that the loss of subjects was related to either baseline lung function or exposure to air pollution. In addition, we observed significant associations between air pollution and lung growth in the subgroup of children who were followed for the full eight years of the study, with effects that were similar in magnitude to those in the group as a whole, thus making loss of subjects an unlikely source of bias.

We have shown that exposure to ambient air pollution is correlated with significant deficits in respiratory growth over an eight-year period, leading to clinically important deficits in lung function at the age of 18 years. The specific pollutants that were associated with these deficits included nitrogen dioxide, acid vapor, $PM_{2.5}$, and elemental carbon. These pollutants are products of primary fuel combustion, and since they are present at similar levels in many other areas,[37,38] we believe that our results can be generalized to children living outside southern California. Given the magnitude of the observed effects and the importance of lung function as a determinant of morbidity and mortality during adulthood, continued emphasis on the identification of strategies for reducing levels of urban air pollutants is warranted.

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REFERENCES

1. Frischer T, Studnicka M, Gartner C, et al. Lung function growth and ambient ozone: a three-year population study in school children. Am J Respir Crit Care Med 1999;160:390-6.
2. Jedrychowski W, Flak E, Mroz E. The adverse effect of low levels of ambient air pollutants on lung function growth in preadolescent children. Environ Health Perspect 1999;107:669-74.
3. Horak F Jr, Studnicka M, Gartner C, et al. Particulate matter and lung function growth in children: a 3-yr follow-up study in Austrian schoolchildren. Eur Respir J 2002; 19:838-45.
4. Gauderman WJ, McConnell R, Gilliland F, et al. Association between air pollution and lung function growth in southern California children. Am J Respir Crit Care Med 2000;162:1383-90.
5. Avol EL, Gauderman WJ, Tan SM, London SJ, Peters JM. Respiratory effects of relocating to areas of differing air pollution levels. Am J Respir Crit Care Med 2001; 164:2067-72.
6. Gauderman WJ, Gilliland GF, Vora H, et al. Association between air pollution and lung function growth in southern California children: results from a second cohort. Am J Respir Crit Care Med 2002;166:76-84.
7. Ware JH, Ferris BG Jr, Dockery DW, Spengler JD, Stram DO, Speizer FE. Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. Am Rev Respir Dis 1986;133: 834-42.
8. Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG Jr. Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 1989;139: 587-94.
9. Schwartz J. Lung function and chronic exposure to air pollution: a cross-sectional analysis of NHANES II. Environ Res 1989; 50:309-21.
10. Raizenne M, Neas LM, Damokosh AI, et al. Health effects of acid aerosols on North American children: pulmonary function. Environ Health Perspect 1996;104:506-14.
11. Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. Health effects of outdoor air pollution. Am J Respir Crit Care Med 1996;153:3-50, 477-98.
12. Peters JM, Avol E, Gauderman WJ, et al. A study of twelve Southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. Am J Respir Crit Care Med 1999;159:768-75.
13. Peters JM, Avol E, Navidi W, et al. A study of twelve Southern California communities with differing levels and types of air pollution. I. Prevalence of respiratory morbidity. Am J Respir Crit Care Med 1999; 159:760-7.
14. Wang X, Dockery DW, Wypij D, et al. Pulmonary function growth velocity in children 6 to 18 years of age. Am Rev Respir Dis 1993;148:1502-8.
15. Elemental carbon (diesel exhaust). In: NIOSH manual of analytical methods. No. 5040. Issue 3 (interim report). Cincinnati: National Institute of Occupational Safety and Health, 1999.
16. SAS/STAT user's guide, version 9. Cary, N.C.: SAS Institute, 2002.
17. Tager IB, Weiss ST, Munoz A, Rosner B, Speizer FE. Longitudinal study of the effects of maternal smoking on pulmonary function in children. N Engl J Med 1983;309: 699-703.
18. Wang X, Wypij D, Gold DR, et al. A longitudinal study of the effects of parental smoking on pulmonary function in children 6-18 years. Am J Respir Crit Care Med 1994; 149:1420-5.
19. Tager I, Munoz A, Rosner B, Weiss ST, Carey V, Speizer FE. Effect of cigarette smoking on the pulmonary function of children and adolescents. Am Rev Respir Dis 1985;131:752-9.
20. Mckean M, Leech M, Lambert PC, Hewitt C, Myint S, Silverman M. A model of viral wheeze in nonasthmatic adults: symptoms and physiology. Eur Respir J 2001;18: 23-32.
21. Schroeder EB, Welch VL, Couper D, et al. Lung function and incident coronary heart disease: the Atherosclerosis Risk in Communities Study. Am J Epidemiol 2003; 158:1171-81.
22. Schunemann HJ, Dorn J, Grant BJ, Winkelstein W Jr, Trevisan M. Pulmonary function is a long-term predictor of mortality in the general population: 29-year follow-up of the Buffalo Health Study. Chest 2000; 118:656-64.
23. Knuiman MW, James AL, Davitini ML, Ryan G, Bartholomew HC, Musk AW. Lung function, respiratory symptoms, and mortality: results from the Busselton Health Study. Ann Epidemiol 1999;9:297-306.
24. Hole DJ, Watt GC, Davey Smith G, Hart CL, Gillis CR, Hawthorne VM. Impaired

lung function and mortality risk in men and women: findings from the Renfrew and Paisley prospective population study. BMJ 1996;313:711-5.
25. Kannell WB, Hubert H, Lew EA. Vital capacity as a predictor of cardiovascular disease: the Framingham Study. Am Heart J 1983;105:311-5.
26. Friedman GD, Klatsky AL, Siegelaub AB. Lung function and risk of myocardial infarction and sudden cardiac death. N Engl J Med 1976;294:1071-5.
27. Ashley F, Kannell WB, Sorlie PD, Masson R. Pulmonary function: relation to aging, cigarette habit, and mortality. Ann Intern Med 1975;82:739-45.
28. Detels R, Tashkin DP, Sayre JW, et al. The UCLA population studies of chronic obstructive respiratory disease. 9. Lung function changes associated with chronic exposure to photochemical oxidants: a cohort study among never-smokers. Chest 1987; 92:594-603.
29. Detels R, Tashkin DP, Sayre JW, et al. The UCLA population studies of CORD. X. A cohort study of changes in respiratory function associated with chronic exposure to SOx, NOx, and hydrocarbons. Am J Public Health 1991;81:350-9.
30. Tashkin DP, Detels R, Simmons M, et al. The UCLA population studies of chronic obstructive respiratory disease. XI. Impact of air pollution and smoking on annual change in forced expiratory volume in one second. Am J Respir Crit Care Med 1994;149:1209-17.
31. Tager IB. Air pollution and lung function growth: is it ozone? Am J Respir Crit Care Med 1999;160:387-9.
32. Avol EL, Navidi WC, Rappaport EB, Peters JM. Acute effects of ambient ozone on asthmatic, wheezy, and healthy children. Res Rep Health Eff Inst 1998;82:1-30.
33. Sarnat JA, Schwartz J, Catalano PJ, Suh HH. Gaseous pollutants in particulate matter epidemiology: confounders or surrogates? Environ Health Perspect 2001;109:1053-61.
34. Ochs M, Nyengaard JR, Jung A, et al. The number of alveoli in the human lung. Am J Respir Crit Care Med 2004;169:120-4.
35. Churg A, Brauer M, del Carmen Avila-Casado M, Fortoul TI, Wright JL. Chronic exposure to high levels of particulate air pollution and small airway remodeling. Environ Health Perspect 2003;111:714-8.
36. Sherwin RP, Richters V, Krafft P, Richters A. Centriacinar region inflammatory disease in young individuals: a comparative study of Miami and Los Angeles residents. Virchows Arch 2000;437:422-8.
37. Tolocka M, Solomon P, Mitchell W, Norris G, Gemmill D, Wiener R. East vs. West in the US: chemical characteristics of PM2.5 during the winter of 1999. Aerosol Sci Technol 2001;34:88-96.
38. Latest findings on national air quality: 2002 status and trends. Research Triangle Park, N.C.: Environmental Protection Agency, 2003. (Report no. 454/K-03-001.)

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Researchers Link Childhood Asthma to Exposure to Traffic-related Pollution

USC investigators show proximity to freeways poses respiratory risk

LOS ANGELES (Sept. 20) —Living near a freeway may mean more than the annoying rumble of cars and trucks: For children, it brings an increased risk of asthma, according to researchers at the Keck School of Medicine of the University of Southern California.

Scientists studying air pollution levels in 10 Southern California cities found that the closer children live to a freeway, the greater their chance of having been diagnosed with asthma. They report their findings in the November issue of the journal *Epidemiology*.

Researchers also found that children who had higher levels of nitrogen dioxide, or NO_2, in the air around their homes were more likely to have developed asthma. NO_2 is a product of pollutants emitted from combustion engines, such as those in cars and trucks.

"These results suggest that tailpipe pollutants from freeway traffic are a significant risk factor for asthma," says lead author James Gauderman, Ph.D., associate professor of preventive medicine at the Keck School. "Considering the enormous costs associated with childhood asthma, today's public policy toward regulating pollutants may merit some re-evaluation."

"These results have both scientific and public health implications," says David A. Schwartz, M.D., director of the National Institute of Environmental Health Sciences, the federal agency that funded the study. "They strengthen an emerging body of evidence that air pollution can cause asthma, and that exposure to outdoor levels of nitrogen dioxide and other traffic-related air pollutants may be a significant risk factor for this illness."

Researchers looked at the pollution-asthma link in 208 children who were part of the USC-led Children's Health Study, the longest investigation ever into air pollution and kids' health. The study has tracked the respiratory health of children in a group of Southern California cities since 1993.

The investigators placed air samplers outside the home of each student to measure NO_2 levels. In addition, they determined the distance of each child's home from local freeways, as well as how many vehicles traveled within 150 meters (about 164 yards) of the child's home. Finally, they estimated traffic-related air pollution levels at each child's

home using models that take weather conditions, vehicle counts and other important factors into account.

-more-

In all, 31 children (15 percent) had asthma. Scientists found a link between asthma prevalence in the children and NO_2 levels at their homes. For each increase of 5.7 parts per billion in average NO_2 – which represents a typical range from low to high pollution levels among Southern California cities – the risk of asthma increased by 83 percent. Risk of wheezing and current asthma medication use also rose as NO_2 levels increased.

They also found that the closer the students lived to a freeway, the higher the NO_2 levels outside their homes. NO_2 levels also corresponded with traffic-related pollution estimates from the group's statistical model.

It was not surprising, then, when they found that the closer the students lived to a freeway, the higher the students' asthma prevalence. For every 1.2 kilometers (about three-quarters of a mile) the students lived closer to the freeway, asthma risk increased by 89 percent. For example, students who lived 400 meters from the freeway had an 89 percent higher risk of asthma than students living 1,600 meters away from the freeway.

Interestingly, the researchers saw that air pollution from freeway traffic influenced NO_2 concentrations at homes more strongly than pollution from other types of roads. Traffic counts within 150 meters of homes (which primarily comprised traffic from smaller streets) were only weakly correlated with measured NO_2.

In any community, a freeway is a major source of air pollution. "Cars and trucks traveling on freeways and other large roads may be a bigger source of pollutants that matter for asthma than traffic on smaller roads," Gauderman says. Scientists also find it difficult to get good data on traffic on smaller streets, which may make it harder to find associations between asthma and local traffic.

Gauderman cautions that researchers do not yet know that NO_2 is to blame for the asthma. NO_2 travels together with other airborne pollutants, such as particulate matter, so it may be a marker for other asthma-causing pollutants.

Study sites included the cities of Alpine, Atascadero, Lake Elsinore, Lancaster, Long Beach, Mira Loma, Riverside, San Dimas, Santa Maria and Upland.

The Children's Health Study is supported by the NIEHS, California Air Resources Board, the Southern California Particle Center and Supersite, the Environmental Protection Agency and the Hastings Foundation.

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W.J. Gauderman, E. Avol, F. Lurmann, N. Kuenzli, F. Gilliland, J. Peters and R. McConnell, "Childhood Asthma and Exposure to Traffic and Nitrogen Dioxide," *Epidemiology*. Vol. 16, No. 6, November 2005.

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Embargoed until Sept. 20, 2005

Contact: Kathleen O'Neil
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Air Pollution Found to Pose Greater Danger to Health than Earlier Thought

USC-led study shows significant death risk linked to airborne particles

LOS ANGELES (Sept. 20) – Experts may be significantly underestimating air pollution's role in causing early death, according to a team of American and Canadian researchers, who studied two decades' worth of data on residents of the Los Angeles metro area.

When the epidemiologists examined links between particle pollution and mortality within more than 260 Los Angeles neighborhoods, they found that pollution's chronic health effects are two to three times greater than earlier believed. The study appears in the November issue of *Epidemiology* but was published early on the journal's Web site.

Among participants, for each increase of 10 micrograms per cubic meter ($\mu g/m^3$) of fine particles in the neighborhood's air, the risk of death from any cause rose by 11 to 17 percent, according to Michael Jerrett, Ph.D., associate professor of preventive medicine at the Keck School of Medicine of the University of Southern California and the paper's lead author. Fine particle levels can differ by about 20 $\mu g/m^3$ from the cleanest parts of Los Angeles to the most polluted.

"By looking at the effects of pollution within communities, not only did we observe pollution's influence on overall mortality, but we saw specific links between particulate matter and death from ischemic heart disease, such as heart attack, as well as lung cancers," Jerrett says. Ischemic heart disease mortality risks rose by 25 to 39 percent for the 10 $\mu g/m^3$ increase in air pollution.

Earlier studies took one or two pollution measures from several cities and compared health effects among cities. This study digs more deeply, taking pollution measures at 23 sites within Los Angeles to more accurately reflect air pollution exposure where residents live and work.

Researchers examined data from 22,906 residents of Los Angeles, Riverside, San Bernardino and Ventura counties in the American Cancer Society's Cancer Prevention Study II since 1982. They determined air pollution exposure in 267 different zip codes where participants lived. The vast number of participants allowed scientists to control for dozens of factors that influence health outcome, such as smoking, diet and education. Finally, they compiled causes of death for the 5,856 participants who died by 2000.

When considering air pollution, the epidemiologists specifically looked at levels of particulate matter, a mixture of airborne microscopic solids and liquid droplets. That includes acids (such as nitrates), organic chemicals, metals, dust and allergens.

-more-

Small particles less than 2.5 micrometers in diameter pose the greatest problems to health because they can penetrate deep into the lungs and sometimes even enter the bloodstream. In this study, the researchers tracked this particulate matter, called $PM_{2.5}$ for short, across the neighborhoods of Los Angeles. It is often found in smoke, vehicle exhaust, industrial emissions and haze, driven by the burning of fossil fuels. Scientists also tracked ozone pollution, but found no link between ozone levels and mortality.

Increased deaths from heart disease jibe with the scientists' earlier research showing links between air pollution and atherosclerosis, a thickening of artery walls that may lead to heart attack and stroke. They believe particulate matter may promote inflammatory processes, including atherosclerosis, in key tissues. "We have convincing evidence that those causes of death that we might expect from inflammation, ischemic heart disease and lung disorders, are elevated in areas of higher pollution levels," he says.

Researchers also saw more than a twofold increased risk of death from diabetes, although numbers of diabetes-related deaths were smaller than those from heart disease, making findings less reliable. "People who are diabetic may be more susceptible to day-to-day fluctuations in air pollution," Jerrett says. "They may experience a state of greater inflammation—related to insulin resistance—that makes their lungs more receptive to receiving harmful particles."

Jerrett notes that findings might have been affected by participants who moved during the study or who changed their lifestyle since 1982. Another limitation is that scientists could only use participants' zip codes, rather than their home addresses, to determine their home neighborhood.

Researchers will conduct a similar study in New York City to try to duplicate findings. They hope to determine whether Los Angeles' tailpipe-emission-driven pollution poses a greater danger than that in the eastern United States, where power plants and factories contribute more heavily to pollution. They also plan to better understand pollution's effects on diabetes, and will use more specific measures to assess pollution within neighborhoods.

Because of the large number of participants in the American Cancer Society's study (more than a million people in 150 cities), policymakers in the past have relied heavily on findings from the study to set the nation's air-quality standards.

"These findings should give us some pause to think about what we need to do as a society," Jerrett says. "Restrictions on tailpipe emissions have gotten tighter, but there are more trucks and cars on the roads and people are driving farther. This study may cause us to reflect on how we use our cars, what cars we drive and whether we can do anything to make tailpipe emissions from all vehicles less harmful to health."

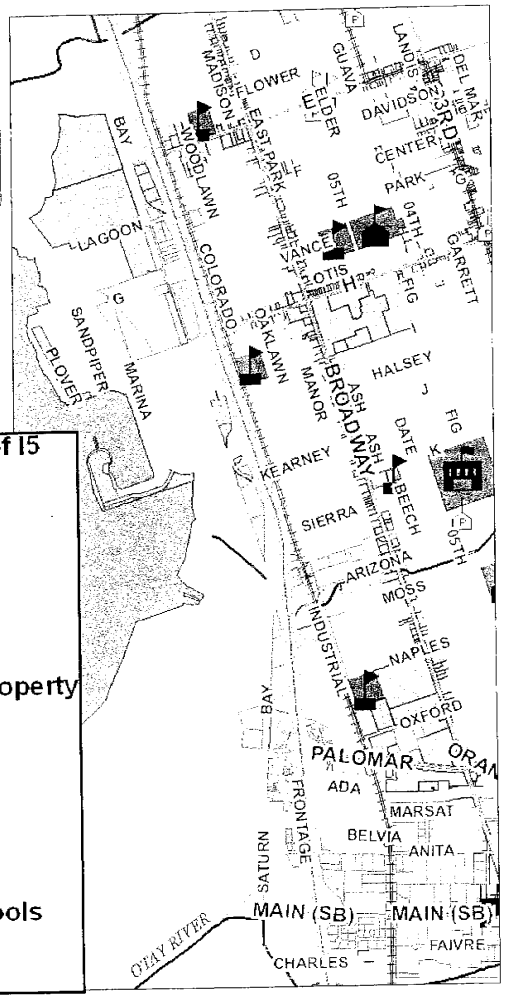
The Health Effects Institute and the National Institute of Environmental Health Sciences supported the research.

# # #

Michael Jerrett, Richard T. Burnett, Renjun Ma, C. Arden Pope III, Daniel Krewski, K. Bruce Newbold, George Thurston, Yuanli Shi, Norm Finkelstein, Eugenia E. Calle and Micheal J. Thun, "Spatial Analysis of Air Pollution and Mortality in Los Angeles," *Epidemiology*. Vol. 16, No. 6 (published early on Epi Fast-Track, www.epidem.com.

For American Cancer Society information, contact David Sampson at (213) 368-8523.

500 Foot Buffer of the I-5 Freeway in West Chula Vista



For more information, please contact Georgette Gomez at Environmental Health Coalition
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[1]California Air Resources Board. Air Quality and Land Use: A Community Health Perspective, April 2005.

BILL NUMBER: **SB 352** CHAPTERED
BILL TEXT

CHAPTER 668
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AMENDED IN ASSEMBLY JULY 16, 2003
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AMENDED IN SENATE MAY 19, 2003
AMENDED IN SENATE MAY 8, 2003
AMENDED IN SENATE MARCH 24, 2003

INTRODUCED BY **Senator Escutia**

FEBRUARY 19, **2003**

An act to amend Section 17213 of the Education Code, and to amend Section 21151.8 of the Public Resources Code, relating to public schools.

LEGISLATIVE COUNSEL'S DIGEST

SB 352, Escutia. Schoolsites: sources of pollution.

Existing law sets forth various requirements regarding the siting, structural integrity, safety, and fitness-for-occupancy of school buildings, including, but not limited to, a prohibition of the approval by the governing board of a school district of the acquisition of a schoolsite by a school district, unless prescribed conditions relating to possible exposure to hazardous substances are satisfied, and a prohibition on the approval of a related environmental impact report or negative declaration.

This bill would, in addition, prohibit the approval by the governing board of a school district of a schoolsite that is within 500 feet from the edge of the closest traffic lane of a freeway or other busy traffic corridor, unless prescribed conditions are met and would make conforming and other technical, nonsubstantive changes.

Existing law requires the lead agency to consult with prescribed agencies to identify facilities that might reasonably be anticipated to emit hazardous materials, within 1/4 of a mile of the schoolsite.

This bill would define "facility" for this purpose and would require the lead agency to consult to identify freeways and other busy traffic corridors, as defined, large agricultural operations, and railyards, within 1/4 of a mile of the schoolsite, and would make conforming and other technical, nonsubstantive changes.

THE PEOPLE OF THE STATE OF CALIFORNIA DO ENACT AS FOLLOWS:

SECTION 1. The Legislature finds and declares all of the following:

(a) Many studies have shown significantly increased levels of pollutants, particularly diesel particulates, in close proximity to freeways and other major diesel sources. A recent study of Los Angeles area freeways measured diesel particulate levels up to 25 times higher near freeways than those levels elsewhere. Much of the pollution from freeways is associated with acute health effects, exacerbating asthma and negatively impacting the ability of children to learn.

(b) Cars and trucks release at least forty different toxic air contaminants, including, but not limited to, diesel particulate, benzene, formaldehyde, 1,3-butadiene and acetaldehyde. Levels of these pollutants are generally concentrated within 500 feet of freeways and very busy roadways.

(c) Current state law governing the siting of schools does not specify whether busy freeways should be included in environmental impact reports of nearby "facilities." Over 150 schools are already estimated to be within 500 feet of extremely high traffic roadways.

(d) A disproportionate number of economically disadvantaged pupils may be attending schools that are close to busy roads, putting them at an increased risk of developing bronchitis from elevated levels of several pollutants associated with traffic. Many studies have confirmed that increased wheezing and bronchitis occurs among children living in high traffic areas.

(e) It is therefore the intent of the Legislature to protect school children from the health risks posed by pollution from heavy freeway traffic and other nonstationary sources in the same way that they are protected from industrial pollution.

SEC. 2. Section 17213 of the Education Code is amended to read:

17213. The governing board of a school district may not approve a project involving the acquisition of a schoolsite by a school district, unless all of the following occur:

(a) The school district, as the lead agency, as defined in Section 21067 of the Public Resources Code, determines that the property purchased or to be built upon is not any of the following:

(1) The site of a current or former hazardous waste disposal site or solid waste disposal site, unless if the site was a former solid waste disposal site, the governing board of the school district concludes that the wastes have been removed.

(2) A hazardous substance release site identified by the Department of Toxic Substances Control in a current list adopted pursuant to Section 25356 of the Health and Safety Code for removal or remedial action pursuant to Chapter 6.8 (commencing with Section 25300) of Division 20 of the Health and Safety Code.

(3) A site that contains one or more pipelines, situated underground or aboveground, that carries hazardous substances, acutely hazardous materials, or hazardous wastes, unless the pipeline is a natural gas line that is used only to supply natural gas to that school or neighborhood.

(b) The school district, as the lead agency, as defined in Section 21067 of the Public Resources Code, in preparing the environmental impact report or negative declaration has consulted with the administering agency in which the proposed schoolsite is located, pursuant to Section 2735.3 of Title 19 of the California Code of

Regulations, and with any air pollution control district or air quality management district having jurisdiction in the area, to identify both permitted and nonpermitted facilities within that district's authority, including, but not limited to, freeways and other busy traffic corridors, large agricultural operations, and railyards, within one-fourth of a mile of the proposed schoolsite, that might reasonably be anticipated to emit hazardous air emissions, or to handle hazardous or acutely hazardous materials, substances, or waste. The school district, as the lead agency, shall include a list of the locations for which information is sought.

(c) The governing board of the school district makes one of the following written findings:

(1) Consultation identified none of the facilities or significant pollution sources specified in subdivision (b).

(2) The facilities or other pollution sources specified in subdivision (b) exist, but one of the following conditions applies:

(A) The health risks from the facilities or other pollution sources do not and will not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the school.

(B) The governing board finds that corrective measures required under an existing order by another governmental entity that has jurisdiction over the facilities or other pollution sources will, before the school is occupied, result in the mitigation of all chronic or accidental hazardous air emissions to levels that do not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school. If the governing board makes this finding, the governing board shall also make a subsequent finding, prior to the occupancy of the school, that the emissions have been mitigated to these levels.

(C) For a schoolsite with a boundary that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor, the governing board of the school district determines, through analysis pursuant to paragraph (2) of subdivision (b) of Section 44360 of the Health and Safety Code, based on appropriate air dispersion modeling, and after considering any potential mitigation measures, that the air quality at the proposed site is such that neither short-term nor long-term exposure poses significant health risks to pupils.

(D) The governing board finds that neither of the conditions set forth in subparagraph (B) or (C) can be met, and the school district is unable to locate an alternative site that is suitable due to a severe shortage of sites that meet the requirements in subdivision (a) of Section 17213. If the governing board makes this finding, the governing board shall adopt a statement of Overriding Considerations pursuant to Section 15093 of Title 14 of the California Code of Regulations.

(d) As used in this section:

(1) "Hazardous air emissions" means emissions into the ambient air of air contaminants that have been identified as a toxic air contaminant by the State Air Resources Board or by the air pollution control officer for the jurisdiction in which the project is located. As determined by the air pollution control officer, hazardous air emissions also means emissions into the ambient air from any substance identified in subdivisions (a) to (f), inclusive, of Section 44321 of the Health and Safety Code.

(2) "Hazardous substance" means any substance defined in Section

25316 of the Health and Safety Code.

(3) "Acutely hazardous material" means any material defined pursuant to subdivision (a) of Section 25532 of the Health and Safety Code.

(4) "Hazardous waste" means any waste defined in Section 25117 of the Health and Safety Code.

(5) "Hazardous waste disposal site" means any site defined in Section 25114 of the Health and Safety Code.

(6) "Administering agency" means any agency designated pursuant to Section 25502 of the Health and Safety Code.

(7) "Handle" means handle as defined in Article 1 (commencing with Section 25500) of Chapter 6.95 of Division 20 of the Health and Safety Code.

(8) "Facilities" means any source with a potential to use, generate, emit or discharge hazardous air pollutants, including, but not limited to, pollutants that meet the definition of a hazardous substance, and whose process or operation is identified as an emission source pursuant to the most recent list of source categories published by the California Air Resources Board.

(9) "Freeway or other busy traffic corridors" means those roadways that, on an average day, have traffic in excess of 50,000 vehicles in a rural area as defined in Section 50101 of the Health and Safety Code, and 100,000 vehicles in an urban area, as defined in Section 50104.7 of the Health and Safety Code.

SEC. 3. Section 21151.8 of the Public Resources Code is amended to read:

21151.8. (a) An environmental impact report or negative declaration may not be approved for any project involving the purchase of a schoolsite or the construction of a new elementary or secondary school by a school district unless all of the following occur:

(1) The environmental impact report or negative declaration includes information that is needed to determine if the property proposed to be purchased, or to be constructed upon, is any of the following:

(A) The site of a current or former hazardous waste disposal site or solid waste disposal site and, if so, whether the wastes have been removed.

(B) A hazardous substance release site identified by the Department of Toxic Substances Control in a current list adopted pursuant to Section 25356 of the Health and Safety Code for removal or remedial action pursuant to Chapter 6.8 (commencing with Section 25300) of Division 20 of the Health and Safety Code.

(C) A site that contains one or more pipelines, situated underground or aboveground, that carries hazardous substances, acutely hazardous materials, or hazardous wastes, unless the pipeline is a natural gas line that is used only to supply natural gas to that school or neighborhood, or other nearby schools.

(D) A site that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor.

(2) The school district, as the lead agency, in preparing the environmental impact report or negative declaration has notified in writing and consulted with the administering agency in which the proposed schoolsite is located, pursuant to Section 2735.3 of Title 19 of the California Code of Regulations, and with any air pollution control district or air quality management district having jurisdiction in the area, to identify both permitted and nonpermitted

facilities within that district's authority, including, but not limited to, freeways and busy traffic corridors, large agricultural operations, and railyards, within one-fourth of a mile of the proposed schoolsite, that might reasonably be anticipated to emit hazardous emissions or handle hazardous or acutely hazardous materials, substances, or waste. The notification by the school district, as the lead agency, shall include a list of the locations for which information is sought.

(3) The governing board of the school district makes one of the following written findings:

(A) Consultation identified no facilities of this type or other significant pollution sources specified in paragraph (2).

(B) The facilities or other pollution sources specified in paragraph (2) exist, but one of the following conditions applies:

(i) The health risks from the facilities or other pollution sources do not and will not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school.

(ii) Corrective measures required under an existing order by another agency having jurisdiction over the facilities or other pollution sources will, before the school is occupied, result in the mitigation of all chronic or accidental hazardous air emissions to levels that do not constitute an actual or potential endangerment of public health to persons who would attend or be employed at the proposed school. If the governing board makes a finding pursuant to this clause, it shall also make a subsequent finding, prior to occupancy of the school, that the emissions have been so mitigated.

(iii) For a schoolsite with a boundary that is within 500 feet of the edge of the closest traffic lane of a freeway or other busy traffic corridor, the governing board of the school district determines, through analysis pursuant to paragraph (2) of subdivision (b) of Section 44360 of the Health and Safety Code, based on appropriate air dispersion modeling, and after considering any potential mitigation measures, that the air quality at the proposed site is such that neither short-term nor long-term exposure poses significant health risks to pupils.

(C) The facilities or other pollution sources specified in paragraph (2) exist, but conditions in clause (i), (ii) or (iii) of subparagraph (B) cannot be met, and the school district is unable to locate an alternative site that is suitable due to a severe shortage of sites that meet the requirements in subdivision (a) of Section 17213 of the Education Code. If the governing board makes this finding, the governing board shall adopt a statement of Overriding Considerations pursuant to Section 15093 of Title 14 of the California Code of Regulations.

(4) Each administering agency, air pollution control district, or air quality management district receiving written notification from a lead agency to identify facilities pursuant to paragraph (2) shall provide the requested information and provide a written response to the lead agency within 30 days of receiving the notification. The environmental impact report or negative declaration shall be conclusively presumed to comply with this section as to the area of responsibility of any agency that does not respond within 30 days.

(b) If a school district, as a lead agency, has carried out the consultation required by paragraph (2) of subdivision (a), the environmental impact report or the negative declaration shall be conclusively presumed to comply with this section, notwithstanding

any failure of the consultation to identify an existing facility or other pollution source specified in paragraph (2) of subdivision (a).

(c) As used in this section and Section 21151.4, the following definitions shall apply:

(1) "Hazardous substance" means any substance defined in Section 25316 of the Health and Safety Code.

(2) "Acutely hazardous material" means any material defined pursuant to subdivision (a) of Section 25532 of the Health and Safety Code.

(3) "Hazardous waste" means any waste defined in Section 25117 of the Health and Safety Code.

(4) "Hazardous waste disposal site" means any site defined in Section 25114 of the Health and Safety Code.

(5) "Hazardous air emissions" means emissions into the ambient air of air contaminants that have been identified as a toxic air contaminant by the State Air Resources Board or by the air pollution control officer for the jurisdiction in which the project is located. As determined by the air pollution control officer, hazardous air emissions also means emissions into the ambient air from any substances identified in subdivisions (a) to (f), inclusive, of Section 44321 of the Health and Safety Code.

(6) "Administering agency" means an agency designated pursuant to Section 25502 of the Health and Safety Code.

(7) "Handle" means handle as defined in Article 1 (commencing with Section 25500) of Chapter 6.95 of Division 20 of the Health and Safety Code.

(8) "Facilities" means any source with a potential to use, generate, emit or discharge hazardous air pollutants, including, but not limited to, pollutants that meet the definition of a hazardous substance, and whose process or operation is identified as an emission source pursuant to the most recent list of source categories published by the California Air Resources Board.

(9) "Freeway or other busy traffic corridors" means those roadways that, on an average day, have traffic in excess of 50,000 vehicles in a rural area, as defined in Section 50101 of the Health and Safety Code, and 100,000 vehicles in an urban area, as defined in Section 50104.7 of the Health and Safety Code.